

# Occipital lobe infarctions are different

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**Objectives:** We hypothesized that occipital lobe infarctions differ from infarctions in other locations as to etiology, risk factors and prognosis among young adults.

**Methods:** Location, etiology, risk factors and long-term outcome were evaluated among all young adults 15–49 years suffering from cerebral infarction in Hordaland County, Norway between 1988 and 1997.

**Results:** The following variables were more frequent among patients with occipital lobe infarction compared with patients with infarctions located elsewhere: younger age ( $P < 0.001$ ), female sex ( $P = 0.016$ ), prothrombotic state ( $P = 0.005$ ) and lack of hypertension ( $P = 0.001$ ). There was no difference as to long-term mortality or recurrence of cerebral infarction.

**Conclusion:** Occipital lobe infarctions differ from infarctions in other locations among young adults. This may have important etiologic and therapeutical implications that need further studies.

**Keywords:** cerebral infarction, occipital lobe, young adults

## Introduction

It has been shown in a number of studies that the distribution of etiology of cerebral infarction among young patients differs from the distribution among older patients (Adams et al 1993; Naess et al 2004). Further, it has been shown that the long-term prognosis among young patients with cerebral infarction differs as to etiology and risk factors (Naess et al 2005). These differences may have important therapeutic implications. It is possible that different locations of the brain are differently susceptible to various etiologies of cerebral infarction. Thus, we hypothesized that infarctions in the occipital lobe differ as to the distribution of etiology, risk factors and the long-term prognosis compared with infarctions located in other parts of the brain.

## Methods

Patients 15–49 years old suffering from a first-ever cerebral infarction from 1988 to 1997 and living in Hordaland County, Norway for at least 5 years before the stroke occurred were included. Cases were found by computer search from hospital registries at each of the five hospitals in the county. The search criteria were patients 15–49 years old admitted to an inpatient or outpatient department of one of the hospitals from 1988 to 1997 (10 years) and discharged with a diagnosis of primary or secondary stroke: categories 430–438 of the *International Classification of Diseases*, ninth revision (ICD-9).

Cerebral infarction was defined in accordance with the Baltimore-Washington Cooperative Young Stroke Study Criteria comprising neurological deficits lasting more than 24 hours because of ischemic lesions, or transient ischemic attacks where computed tomography (CT) or magnetic resonance imaging (MRI) showed infarctions related to the clinical findings (Johnson et al 1995). We excluded patients with cerebral infarction associated with other intracranial diseases such as subarachnoidal hemorrhage, sinus venous thrombosis, or severe head trauma.

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Hypertension was defined as treatment with antihypertensive drugs before stroke onset or the introduction of antihypertensive treatment before discharge because of repeated blood pressure measurements  $>140/90$  mmHg. Diabetes mellitus was considered present if it was diagnosed before stroke onset (patient on glucose-lowering diet or medication) or during the hospital (fasting plasma glucose  $>7.7$  mmol/L several days after stroke onset). Current smoking at stroke onset was defined as smoking at least one cigarette per day. Angina pectoris and myocardial infarction were considered present if diagnosed by a physician any time before stroke onset. The presence of migraine was based on self-assessment. The diagnostic work-up comprised CT, MRI, electrocardiography, echocardiography, Doppler sonography of extra- and intra-cranial arteries, conventional angiography, and laboratory studies including complete blood cell count, electrolytes, creatinine, glucose, cholesterol, protein C, protein S, antithrombin III, anticardiolipin antibodies, lupus anticoagulant, and homocysteine. Causation was based on the TOAST (Trial of Org 10172 in Acute Stroke Treatment) criteria (Adams et al 1993).

All surviving patients were invited to a follow-up visit. Information on recurrence of cerebral infarction and post-stroke myocardial infarction was based on self-report and review of all patient records. Blood samples were drawn at follow-up for determination of total cholesterol, high-density lipoprotein (HDL), and low-density lipoprotein (LDL). A total of 199 (95.2%) of all surviving patients came to the follow-up control.

## Statistics

Independent samples T-test and Fisher's exact test were used when appropriate. Multivariate analysis was made using occipital lobe infarction (OLI) or extra-occipital lobe infarction (ELI) as dependent variable. The analyses were performed with SPSS 11.0.1 for Windows.

## Results

The total patient population comprised 136 men (59%) and 96 women (41%). Nineteen (8.2%) patients had OLI and 213 (91.8%) patients had ELI. Table 1 shows the demography of OLI and ELI patients. OLI patients were younger ( $P < 0.001$ ) and more frequent female ( $P = 0.016$ ). None of the OLI patients had hypertension ( $P = 0.001$ ). There were no differences as to diabetes mellitus, smoking, migraine and coronary heart disease. Systolic and diastolic blood pressures on admittance were significantly lower among the OLI patients. CT was negative among 39.6% ELI patients and 26.3% OLI patients ( $P = 0.497$ ). A prothrombotic state was present among 26.3% OLI patients

**Table 1** Demography of young adults with occipital infarctions compared with infarctions other than occipital

	Occipital infarctions %	Other infarctions %	P
Male	31.6	60.8	0.016
Mean age (years)	33.5	42.3	$<0.001$
Small-artery occlusion	0	16.0	ns
Dissection	0	6.6	ns
Prothrombotic state	26.3	5.2	0.005
Large-artery atherosclerosis	10.5	15.6	ns
Cardioembolism	10.5	7.5	ns
Stroke of other determined cause	5.3	1.4	ns
Stroke of undetermined cause	52.6	47.6	ns
Hypertension	0	34.4	0.001
Diabetes mellitus	10.5	11.8	ns
Smoking	47.4	38.2	ns
Migraine	15.8	16.6	ns
Myocardial infarction prestroke	5.3	7.5	ns
Angina pectoris prestroke	0	7.5	ns
Mean systolic blood pressure on admittance (mmHg)	126	149	0.020
Mean diastolic blood pressure on admittance (mmHg)	74	92	0.002
Total cholesterol on follow-up (mmol/L)	5.2	6.1	0.003
LDL on follow-up (mmol/L)	3.0	4.0	0.001
HDL on follow-up (mmol/L)	1.4	1.3	ns

**Abbreviations:** HDL, high-density lipoprotein; LDL, low-density lipoprotein; ns, not significant.

and 5.2% ELI patients ( $P = 0.005$ ). Among OLI patients the prothrombotic state included pregnancy ( $n = 2$ ), antithrombin III deficiency ( $n = 2$ ), and polycythemia vera ( $n = 1$ ). Among ELI patients the prothrombotic state included pregnancy ( $n = 2$ ), post-partum state ( $n = 2$ ), protein S deficiency ( $n = 2$ ), lupus anticoagulant ( $n = 1$ ), thrombocytopenic purpura ( $n = 1$ ), and systemic lupus erythematosus ( $n = 1$ ). The only OLI patient with stroke of other determined cause had mitochondrial encephalomyopathy, lactic acidosis, and stroke-like episodes (MELAS). Stroke of other determined cause among 3 ELI patients included homocysteinemia, Conn's syndrome and Borrelia-related arteritis.

The mean time to follow-up was 6 years. Twenty-three (9.9%) patients had died. Among ELI patients 9.8% had died while 10.5% OLI patients had died ( $P = 0.857$ ). Recurrence

of ischemic stroke occurred among 9.5% ELI patients and 10.5% OLI patients ( $P = 0.916$ ). Post-stroke myocardial infarction occurred among 11.2% ELI patients and 10.5% OLI ( $P = 1.000$ ). On follow-up, OLI patients had significantly lower total cholesterol and LDL than ELI patients.

Multivariate analysis showed that OLI was associated with low age (odds ratio [OR] = 1.1,  $P = 0.014$ ), low systolic blood pressure on admission for the index stroke (OR = 1.1,  $P = 0.041$ ) and low total-cholesterol on follow-up (OR = 2.0,  $P = 0.066$ ).

## Discussion

The main finding in the present study was that there are important differences at group level between young OLI and ELI patients. Occipital infarction was associated with younger patients and female sex. A prothrombotic state was more frequent among OLI than ELI patients. Furthermore, no OLI patients had hypertension and total cholesterol was lower. This suggests that the distribution of causation of occipital infarction may be different from cerebral infarctions located elsewhere.

The high frequency of a prothrombotic state among OLI patients in the present study is noteworthy. Migrainous infarction is associated with occipital location according to some studies (Hoekstra-van Dalen et al 1996), and plasma hypercoagulability may be important in migraine-related cerebral infarction (Riddle et al 1989). However, the frequency of migraine was similar among OLI and ELI patients in our study. Thus, we found no support for the association between migraine and occipital location of cerebral infarction and it is likely that the high frequency of prothrombotic states among OLI patients was unrelated to migraine. A limitation of the present study was that it was not possible to attribute causation to migraine because the patient records did not provide the information required by the defining criteria of migrainous infarction. Furthermore, the frequency of migraine with aura is unknown in the present study.

On multivariate analysis female sex, prothrombotic state and hypertension were no longer associated with OLI while there was a trend for low age, low systolic blood pressure on admission for the index stroke and low total cholesterol on follow-up to be independently associated with OLI. Although low numbers make multivariate analysis difficult to interpret in the present study, the results support the suggestion that the distribution of causation in OLI and ELI is different. Low total cholesterol suggests that atherosclerosis may be less frequent among OLI patients.

It has been shown that mitochondrial diseases such as MELAS are associated with infarction in the posterior regions of the brain. Except one OLI patient with known MELAS,

our patients were not tested for mitochondrial disease. However, our study raises the possibility that mitochondrial disease may be an important cause of occipital infarctions among young patients. Genetic analyses for mitochondrial diseases should be performed among patients with occipital infarctions in future studies.

We did not find that the prognosis as to mortality, recurrence of cerebral infarction and post-stroke myocardial infarction was different among OLI and ELI patients. A larger number of patients is likely needed to disclose whether occipital lobe infarctions are associated with better prognosis compared with infarctions located elsewhere.

The strength of the present study is its population-based design. It allows unbiased determination of the distribution of stroke location. In comparison, most studies of young ischemic stroke patients are hospital based and thus liable to selection bias.

A limitation is that case-finding was retrospective, which may affect both case-finding and case ascertainment. Nevertheless, the admission rate is high in Norway, and others have found that intensified case-finding efforts among general practitioners in Norway did not significantly increase the incidence rates of stroke (Ellekjaer et al 1997). This indicates that few cases escaped our attention.

In conclusion, our study disclosed occipital infarctions to differ from infarctions in other locations. This may have important etiologic and therapeutical implications that need further studies.

## Acknowledgment and funding

This study was supported by Frank Mohn Foundation, Bergen, Norway.

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